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ADAPTATION TO BREATH-HOLD DIVING

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During the training of submarine crews in submarine escape procedures, such as "free or buoyant ascent," instructors at the escape training tank frequently hold their breath under water and perform "skin" dives to depths as great as 90 feet. The ascent is carried out by climbing up a line. These diving maneuvers are similar to those practiced by sponge and pearl divers. The escape training tank at the New London Submarine Base (Fig. 1) afforded us an opportunity to study the pulmonary gas exchange during this type of diving[8], and to follow up physiological changes in the tank instructors during their tour of duty.

Since the adaptive processes in breath-hold diving observed under these conditions are related to the particular stress of diving imposed on tank instructors, data on alveolar PCO_2 and PO_2 obtained at the end of dives are presented in Table I to serve as a frame of reference. A reversed CO_2 gradient was established during descent[8]. At a 90-foot depth, about 50 per cent of the predive CO_2 content of the lungs had disappeard and was taken up by the blood and tissues.

During ascent the CO_2 gradient is again normal. The influx of carbon dioxide into the lungs can be regulated by the speed of ascent, as shown in Table I. If the ascent was fast, the alveolar CO_2 tension attained on reaching the surface was low, while considerably higher CO_2 tensions were found following slow ascents. It is important to note that extremely low end dive alveolar PO_2 levels (25-30 mm Hg PO_2) are not uncommon, indicating the existing danger of hypoxia in breath-hold dives.

The processes involved in pulmonary gas exchange during the dive, (1) transfer of CO_2 from the lungs, (2) oxygen utilization, and (3) nitrogen transfer into the blood, act in the same direction as the mechanical compression of the thorax during the descent of the diver and cause a progressive shrinking of the total chest volume during the descent. The maximal depth a diver can reach is dependent upon his lung volumes, in particular upon the ratio of total lung volume. In the following, data are presented which indicate that under the conditions at an escape training tank, involving multiple daily breath-hold dives, adaptive changes develop into lung volumes and in the responses to high CO_2 and to low O_2.

Adaptive Changes in Lung Volumes

During their first breath-hold dives, new personnel assigned to the escape training tank usually experience a pressure and stress on their chest at a depth of 60 to 70 feet which prevents them from venturing deeper. After several months they report that they can inspire more air, are able to control their breathing more regularly and are more relaxed. Most of them can eventually reach a depth of 90 feet during breath-hold dives without experiencing any difficulties.

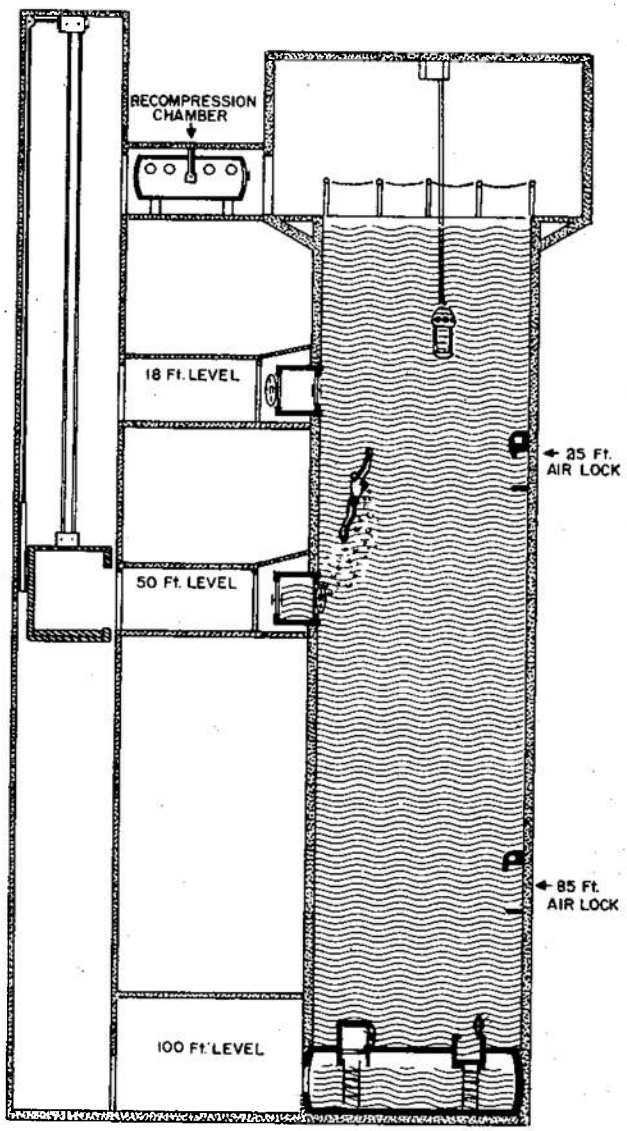

Figure 1. Schematic Diagram of Submarine Escape Training Tower.

TABLE I

End-Dive Alveolar PCO_2 and PO_2 Values Following Descent to 90 Feet and Ascent with Different Speeds

A. Dives to 90 feet Speed of ascent	Alveolar Gas Tensions PCO_2 mm Hg	 PO_2 mm Hg
1.9 ft/sec	45.7 ± 4.1 (12)	34.8 ± 5.7 (12)
2.3 ft/sec	37.3 ± 1.2 (3)	34.7 ± 4.2 (3)
3.5 ft/sec	31.5 ± 1.3 (3)	27.3 ± 3.1 (3)
B. Breath-holding at surface	52.4 ± 2.4 (12)	64.8 ± 13.4 (12)

( ) Number of dives.
Breath-holding at the surface was carried out by the same subjects who performed the dives to 90 feet and ascended at an average speed of 1.9 ft/sec. Breath-holding time of 1.5 minutes corresponded with the average time of breath-hold dives.

These subjective experiences suggested an adaptation of lung volumes, which was established in subsequent studies[2]. A comparison of lung volumes measured in 16 tank instructors and 16 laboratory personnel showed a significantly larger vital capacity in tank instructors, which was 20 per cent higher than could be predicted by their own height, weight, and age, using the West formula[2]. Furthermore, total lung capacity, tidal volume, and inspiratory reserve volume were markedly increased in the tank-instructor group as compared with the laboratory personnel. In a longitudinal study, lung volumes were measured in tank instructors at the beginning of their tour of duty and after one year. Inspiratory reserve, tidal volume, vital capacity and total lung capacity showed a significant increase in 20 tank instructors, while residual volume decreased. In a second group of eight tank instructors, similar studies were carried out and the ratio of total lung capacity to residual volume plus the volume of the airways was determined (Table II). The volume of the airways (anatomical deadspace) was estimated, using Radford's nomogram[6]. The observed change in this ratio results in a 20- to 30-foot extension in the maximum safe depth to which the instructors could dive after one year of duty.

TABLE II

Effect of Prolonged Diving Training (1 Year) on Maximal Attainable Depth Based on Ratio of Total Lung Capacity to Residual Volume and Volume of Airways (8 Divers)

	Beginning	After 1 Year of Duty
	Volume in	ML (BTPS)
Residual Volume	1918 ±510	1621 ±287
Anatomical dead space (based on Radford's nomogram)	172 ±8	169 ±9
Total lung capacity	7373 ±961	7643 888
Ratio total lung capacity/ residual volume and anatomical dead space	3.65 ±.57	4.38 ±.97
Maximal attainable depth (feet	87	112

Adaptation to Increased CO_2 in Divers

CO_2 tolerance curves were obtained by exposing subjects for 15 minutes to 3.3, 5.4 and 7.5 per cent CO_2. Alveolar ventilation and alveolar gas tensions were determined at the end of each exposure period. In the case of tank instructors, the CO_2 tolerance curves showed a shift to the right and a decrease slope when compared with those of the laboratory personnel[9]. The high tolerance to CO_2 is developed during the diving period and lost after a three-month lay-off period, as shown in CO_2 sensitivity tests of eight tank instructors (Fig. 2). The ventilatory response to 5 per cent CO_2 is significantly larger at the end of the three-month lay-off period. The changes in lung volumes, consisting of an increase in total lung capacity, vital capacity and tidal volume, and decrease in residual volume, might contribute to the reduced sensitivity to CO_2 because of the relationship found between large tidal volume, low respiratory rate and low response to CO_2[10].

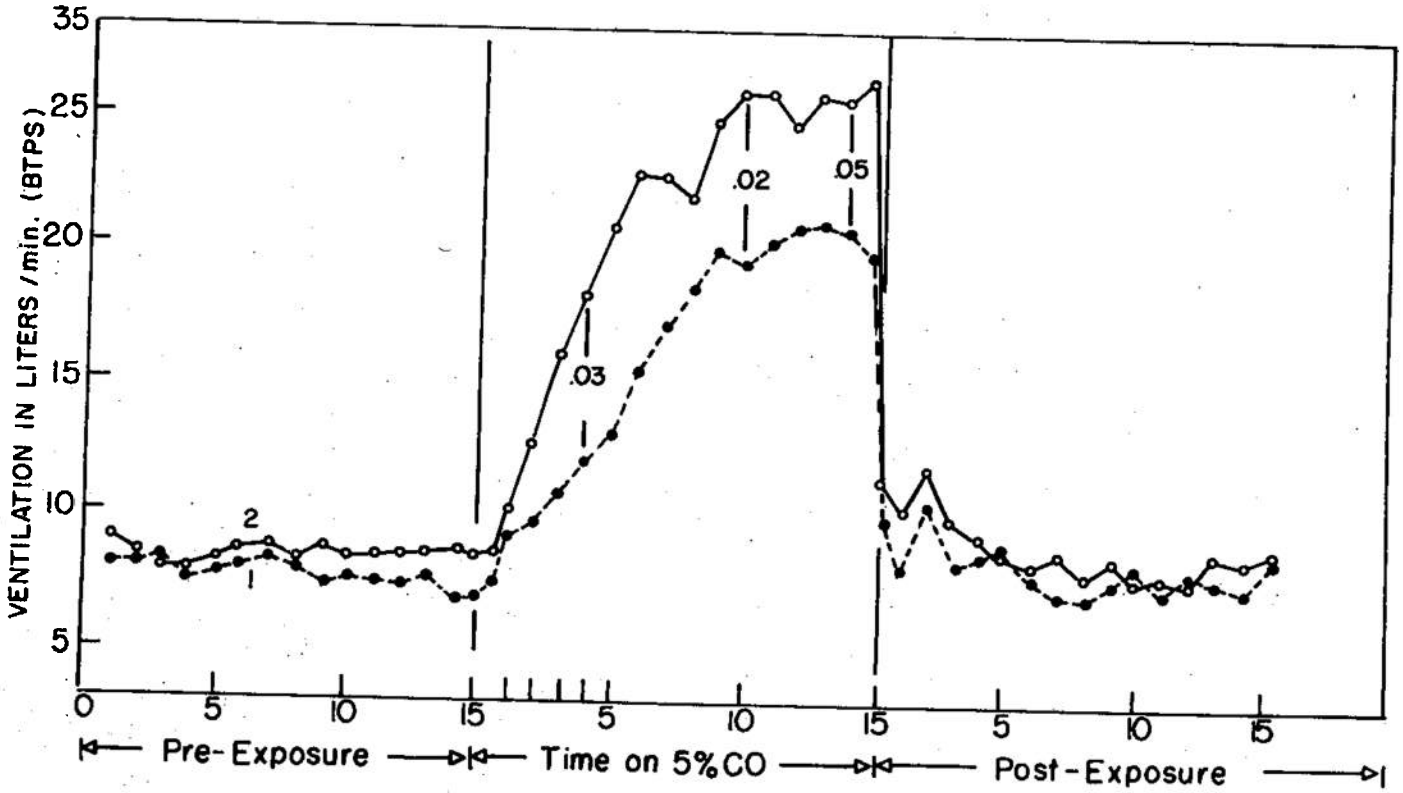

Figure 2. Respiratory Response to 5 per cent CO_2 in Tank Instructors (7 subjects).
(1) During a period of intensive water work.
(2) After a 3-month lay-off period.

Other parameters of adaptation to CO_2 have been previously established in human subjects during prolonged exposure to 1.5 per cent CO_2[11]. They consisted, besides changes in acid base equilibria, in an increase in red cell sodium and a decrease in red cell potassium. Tables III and IV show the distribution of CO_2 in plasma and cells and the distribution of electrolytes in plasma and red cells of 11 tank instructors after a period of heavy water work and a period without water work. It can be seen that after a period of intensive diving, the pH is decreased, PCO_2 and bicarbonate levels are increased, and the sodium and potassium concentrations in red cells exhibit the typical changes observed in prolonged exposure to CO_2. These data provide further evidence of an adaptation to CO_2 during breath-hold diving. Evidence of an increase in CO_2 stores, as a result of diving, was recently obtained in instructors, following a two-year period of water work when compared with data found after a three-month lay-off period[3]. During constant hyperventilation, lasting for one hour, more CO_2 was eliminated and the end tidal CO_2 tension was significantly elevated under the first condition.

Adaptation to Low Oxygen

A group of tank instructors and a group of laboratory personnel were exposed to 10.5 per cent O_2 in nitrogen for a period of 33 minutes.[1] The subjects reported in the morning under basal conditions (14 hours without food) and rested in bed for 45 minutes before the experiment began. They were in supine position and breathed from an open spirometer system through a mouthpiece and exhaled into Douglas bags for an initial period of 33 minutes on room air, followed by 33 minutes on 10.5 per cent O_2 in N_2, and finally a recovery period of 33 minutes on room air. End tidal oxygen and carbon dioxide samples were obtained with a Rahn sampler. Both end tidal gas samples and expired gas samples were analyzed with a Haldane apparatus. Two tests of this type were conducted on the same subjects at an interval of several months.

Table V shows the average data of alveolar oxygen and carbon dioxide pressures, the ventilatory response and the arterial oxygen saturation and pulse rate obtained during the periods of 23 to 33 minutes while breathing room air, 10.5 per cent O_2 in N_2, and again room air in two tests. The ventilatory response of the tank instructors to low oxygen is consistently lower than that of the laboratory personnel while alveolar oxygen and carbon dioxide tensions, as well as the arterial oxygen saturation and pulse rate, do not exhibit marked differences between the two groups.

The lower ventilatory response to low oxygen breathing in the divers was found to be associated with the formation of a larger oxygen debt during exposure. Table VI summarizes the data on oxygen debt, oxygen balance and the end of exposure, compensatory oxygen uptake during the recovery period, final oxygen

[1] The study of low oxygen breathing had originally been carried out on 11 subjects (8). For the purpose of comparison of the low oxygen effects in skin divers and laboratory personnel, three subjects were excluded. One subject had emphysema, another was a hard-hat diver without training in skin diving and a third subject was assigned to the Escape Training Tank and performed, mainly, desk work probably because of maladjustment to skin diving.

TABLE III

Effect of Daily Breath-Hold Dives During a Six-month Period on Distribution of CO_2 in Plasma and Red Cells (venous blood) of 11 Tank Instructors

Plasma				Red Cells	
HCO_3 mmoles/liter	H_2CO_3 mmoles/liter	pH	PCO_2 mm Hg	HCO_3 mmoles/liter	H_2CO_3 mmoles/liter
After a five-month period without water work (control					
25.1 ± 1.5	1.34 ± .09	7.38 ± .01	44.7 ± 2.96	16.76 ± .87	1.12 ± .07
After a six-month period with heavy water work					
28.3* ± 1.38	1,58* ± .18	7.35* + .05	52.7* ± 6.1	18.60* ± .67	1.32 ± .15

*Differences from controls statistically significant at the 5 per cent level.

TABLE IV

Effect of Daily Breath-Hold Dives for a Period of Six Months on Red Cell and Plasma Electrolytes (venous blood) (11 subjects)

Measured Values									Calculated Values			
Whole Blood					Plasma				Red Cells			
H_2O, g/l	Na, mEq/l	K, mEq/l	Cl, mEq/l	Hematocrit	H_2O g/l	Na, mEq/l	K, mEq/l	Cl, mEq/l	H_2O, g/l	Na, mEq/l	K, mEq/l	Cl, mEq/l
After a five-month period without water work (control)												
824 ±12.8	86.8 ±5.0	43.9 ±2.3	84.5 ±2.3	43.0 ±1.9	924 ±7.3	142 ±3.5	4.78 ±.62	103.5 ±3.0	692 ±27	13.7 ±3.8	95.8 ±15.4	59.2 ±6.6
After a six-month period with heavy water work												
811* ±5	86.8 ±2.9	34.6* ±2.4	83.5 ±2.4	44.8 ±2.3	915** ±9.3	133* ±4.4	4.09** ±.39	103.5 ±1.6	679 20	30.4** ±17.3	72.1* ±5.4	59.4 ±6.9

*Differences from controls statistically significant at the one per cent level and better.
**Differences from controls statistically significant at the five per cent level.

TABLE V

Average Data of Alveolar Oxygen and Carbon Dioxide Pressures, Ventilatory Response, Pulse Rate and Arterial Oxygen Saturation During the Period of 23-33 Min on Air, 10.5 Per Cent O_2 in N_2 and Air (Recovery)

	Laboratory Personnel (4 Subjects)			Divers (4 Subjects)		
Alveolar mmHg PCO_2	Air Control	10.5% O_2 in N_2	Air Recovery	Air Control	10.5% O_2 in N_2	Air Recovery
1. Test	38.0 ±2.0	32.4 ±5.5	37.3 ±2.6	38.5 ±3.5	32.6 ±4.2	37.9 ±3.0
2. Test	38.2 ±3.6	31.8 ±5.0	35.8 ±3.3	37.6 ±3.3	33.3 ±3.2	35.4 ±4.4
Alveolar mmHg PO_2	107.5	42.5	109.0	104.9	49.1	101.5
1. Test	±4.3	±5.3	±10.5	4.9	±17.2	±5.9
2. Test	107.9 ±4.6	43.9 ±6.0	107.9 ±6.8	105.4 ±4.6	43.5 ±7.5	106.3 ±7.0
$\dot{V}E$ L/min	5.66	9.03	5.82	6.20	7.67	6 00
1. Test	±0.74	±2.11	±1.04	±0.67	±0.66	±0.66
2. Test	5.72 ±0.49	8.71 ±2.0	6.51 ±1.81	5.98 ±0.93	7.53 ±1.22	5.96 ±1.20
$\dot{V}E$, %	100	161	105	100	123	96
1. Test		±13	±4		±10	±5
2. Test	100	152 ±26	100 6	100	123 ±7	98 ±3
Pulse rate	64	79	61	56	74	58
1. Test	±9	±8	±10	±5	±2	±11
2. Test	67 ±8	79 ±12	66 ±13	59 ±11	69 14	53 ±13
Pulse rate %	100	124	96	100	132	99
1. Test		±8	±3		±29	±4
2. Test	100	117 ±7	96 ±5	100	129 ±16	98 ±4
Arterial O_2 Saturation	96	74	96	95	64	96
1. Test	±2	±3	±6	±1	±5	±1
2. Test	96 ±1	74 ±10	97 ±1	95 ±2	74 ±2	97 ±1

TABLE VI

Oxygen Debt and Excess O_2 Uptake, Resulting from 33 Minutes Exposure to 10.5 Per Cent O_2 In N_2, Related Changes in Arterial O_2 Saturation and Oxygen Removal from Inspired Gas, Slope of Oxygen Dissociation Curves in Divers and Non-Divers

	O_2 debt	O_2 balance at 33 min exposure	Excess O_2 uptake above basic values	Final O_2 balance (33 min recovery)	Arterial O_2 saturation (23-33 min exposure)	O_2 removal from inspired gas during 23-33 min exposure to 10.5%O_2	Slope of steepest part of O_2 dissociation curve
	(ml)	(ml)	(ml)	(ml)	(%HbO_2)	(in % O_2)	
Group A							
Laboratory personnel	-347.8	56.3	1204.8	1261	73.8	3.72	.295
4 subjects	±110.6	±338	±851	±838	±11.1	±.66	±.047
Group B							
Divers	-1033.8	-966.5	868.5	-98	74.0	4.26	.388
4 Subjects	±499	±522.7	±536	±823	±2.2	±.211	±.022

balance at the end of the recovery period, oxygen removal from inspired air and the slope of the steepest part of the oxygen dissociation curve. The smaller oxygen debt, incurred by the laboratory personnel during the first 11 minutes of exposure to low oxygen, can be explained by the reduction in O_2 uptake due to the fall in arterial oxygen saturation. Following a suggestion of Dr. H. Rahn, we calculated the reduction in oxygen uptake from the difference in arterial oxygen content while breathing air and 10.5 per cent O_2 and assuming that the A-V difference is unaltered on breathing 10.5 per cent O_2. We further assumed a total circulating blood volume of seven liters. Substituting our value of 22 per cent drop in HbO_2, and converting it to ml/liter — we then have (200-156) x 7 = 308 ml. In addition, there must be a reduction in dissolved oxygen in tissue fluids. Using the mean capillary O_2 as an index of mean tissue PO_2 tension, this value would not change more than 20 to 30 mm Hg upon going to 10.5 per cent O_2 from air. Therefore, assuming a 20 mm Hg drop in this factor with a solubility factor of 0.02 for O_2 in all tissues, this would yield an additional 70 ml of O_2, by which the oxygen uptake would be reduced (calculated for a 70 kg man). These are at best only approximations, but they come close to the average values of the oxygen debt obtained in laboratory personnel (378 ml calculated, 348 ml observed). This oxygen deficit can be met by the oxygen reserves of the organism which can be estimated to be in the order of 1300 ml (including the oxygen content of myoglobin).

It is interesting to note that a 22 per cent drop in arterial oxygen saturation is quantitatively compensated by a 50-60 per cent increase in ventilation and a 20 per cent increase in pulse rate, providing the necessary increase in oxygen supply to the alveoli and increased transportation of blood to overcome the reduction in oxygen uptake. The larger oxygen debt incurred by the divers, over the entire exposure period to 10.5 per cent O_2 cannot be explained by the reduction in oxygen uptake due to the fall in arterial oxygen saturation, although the divers show a slightly better utilization of available oxygen as indicated in the larger differences between inspired and expired oxygen concentrations and a slightly larger slope in the steepest part of the oxygen dissociation curves. The oxygen stores of the organism (1300 ml) which cannot be utilized to the full extent will not be sufficient to meet these larger oxygen debts of the divers which, in one case, was 1562 ml. The divers reach a stable level of ventilatory and circulatory response after the 11th minute of exposure, but continue to accumulate an oxygen debt as exposure to low oxygen proceeds. They do not compensate for the reduced oxygen uptake by an adequate increase in ventilation and pulse rate. We, therefore, must assume that a reduction in tissue oxidation has occurred in the divers.

As indicated above, the oxygen debt in one diver was 1562 ml at the end of the 33 minutes of exposure to 10.5 per cent O_2 and still over 1000 ml at the end of a 33 minute recovery period on air. This same subject showed similar trends in a separate experiment, breathing 15 per cent O_2 in N_2 for 33 minutes during which period he accepted an oxygen debt of 930 ml, and at the end of the 33 minute period of recovery on air it was 430 ml. This finding seems to indicate that the reduction in oxidation is still effective in the recovery period on air following exposure to low oxygen or that a shift to anaerobic energy yielding processes might have occurred..

Damping Effect on the Autonomic Nervous System

It had been previously reported that a high tolerance to inhalation of increased CO_2 concentrations (low ventilatory response) was associated with a reduced autonomic response as indicated in smaller elevations of pulse rate and blood sugar[10]. Most of the subjects belonging to the group showing a high tolerance to CO_2 in the earlier studies[10] were divers. They also did not exhibit marked symptoms during and after CO_2 inhalation in contrast to subjects with a high ventilatory response to CO_2.

Since carbon dioxide exposure is known to produce an increased sympathico-adrenal discharge in men, as seen by a rise of epinephrine, norepinephrine and 17-hydroxy cortico steroids[18], a high tolerance to CO_2, as developed by the divers, appears to be associated with a reduced adrenergic and stress response to CO_2. The question arose, whether adaptation to diving involves a general damping of the autonomic nervous system activity, including the cholinergic system. We measured the blood pressure response to an injection of a cholinergic drug, Mecholyl, in a group of 13 divers and 19 laboratory personnel. Mecholyl (10 mg per ml) was given intramuscularly and the systolic blood pressure was followed every minute for 6 minutes and then at 10, 15, 20 and 25 minutes. The results expressed in per cent change of baseline values are presented in Figure 3. The divers exhibited a significantly smaller fall in blood pressure than the group of laboratory personnel and practically no overshoot above control levels in the period between 15 and 25 minutes following injection. These findings suggest that adaptation to diving also produces a damping effect on the cholinergic system.

Discussion and Conclusion

Comparison of lung volumes in divers and control groups demonstrated a larger vital capacity in the native diving women in Japan[20] and in Korea[19] due to an increased inspiratory reserve volume[19]. Similar observations were made in divers at the escape training tank[2]. These findings suggested an adaptation of lung volumes in breath-hold diving, the existence of which was proven in a longitudinal study in the same divers tested at the beginning and at the end of a long tour of duty at the escape training tank[2].

A lower response to CO_2 in divers, as compared with a native group, has also been reported in the Amas[19] and in tank instructors[9]. Evidence for the development of adaptation to CO_2 during a period of regular daily breath-hold dives has been presented in this report. It appears likely that the adaptive change in lung volumes is related to the decreased respiratory response to CO_2 because of the reported correlations of larger lung volumes and low ventilatory response to CO_2[10].

Experiments with breathing low O_2 mixtures demonstrated that divers are apparently able to utilize oxygen better than non-divers, as indicated in a larger oxygen extraction from inspired gas, and a steeper slope of the oxygen dissociation curve. This may be an effect of adaptation to diving and could offer an explanation for the lower ventilatory response to low O_2 breathing in dives, if it were not associated with a significantly larger oxygen debt. The oxygen debt of the control group could be predicted on the basis of the reduction in O_2 uptake, due to the fall

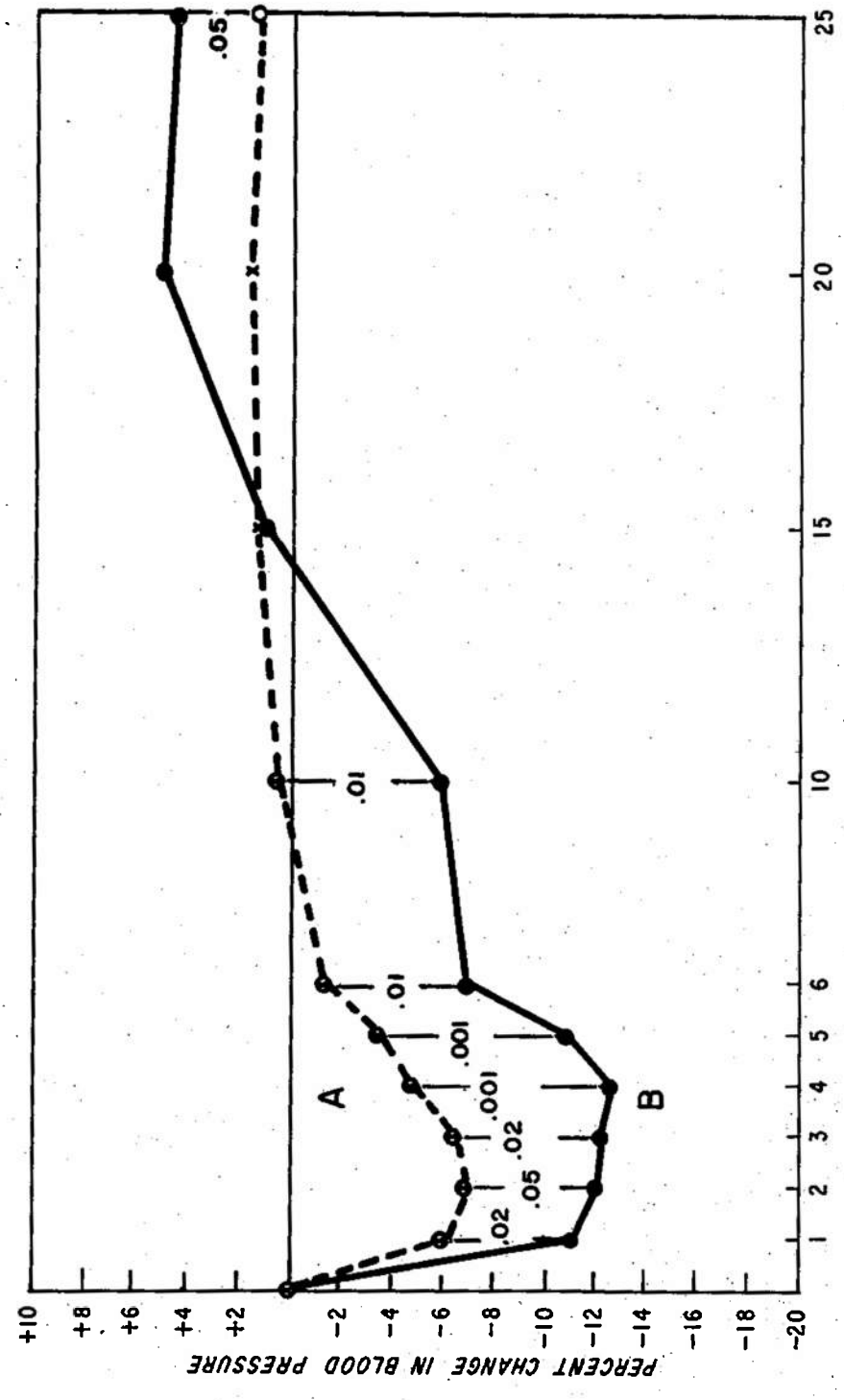

Figure 3. Blood Pressure Response to Mecholyl Injection in 13 Divers (A) and 19 Laboratory Personnel (B)

in HbO_2, on going from air to low O_2 breathing. However, the three-fold larger oxygen debt of the divers, which cannot be met by the oxygen reserves of the organism, requires the assumption of a decreased tissue oxidation. A reduction in energy metabolism has been demonstrated during dives of ducks[1] and seals[15]. The excess oxygen intake of these animals, during the recovery after a dive, covers only a part of the oxygen debt which would have been incurred had the energy metabolism remained at the pre-dive level. They also show a steady temperature loss during breath-hold dives even though they are submerged in thermoneutral water [1, 15].

Since the blood flow through the periphery is reduced during the dive, providing protection against heat loss, it was concluded that the fall of temperature during the dives must have been caused by a decrease in heat production[1, 15, 16]. Scholander[16] also pointed out that the anaerobic energy metabolism during the dive, as measured in the lactic acid production, is too small to compensate for the oxygen deficit under these conditions. However, this does exclude the possibility that other anaerobic energy yielding processes may play a role in diving.

The lowered ventilatory response to low O_2 breathing, found in four experienced and efficient divers, should be confirmed in a larger group of subjects in a longitudinal study, particularly since similar tests in Korean diving women did not show an altered sensitivity to low O_2[19]. The Amas, however, dive to shallower depths than the tank instructors, which might be one of the reasons for the difference observed.

Under the conditions of diving, man is subjected to hypercapnia and hypoxia, and the acclimatization processes are different from those observed during exposure to high altitude, which produces a chronic hypocapnia and chronic hypoxia[4]. At high altitude, the sensitivity to CO_2 is increased, while the sensitivity of O_2 is unaltered[7, 5].

The reduced autonomic responsiveness found in divers is probably a consequence of their adaptation to increased carbon dioxide. The two phases of uncompensated and compensated respiratory acidosis, observed in chronic CO_2 exposure, are associated with a period of excitation followed by a period of depression of the central nervous system[13]. During acute exposure to CO_2 stimulating effects on the hypothalamic cortical system are exerted commensurate with depressing effects on the cerebral cortex. After adaptation to carbon dioxide, which is accomplished with the compensation of the respiratory acidosis, the stimulatory effects of CO_2 on the autonomic system subside and the cortical depressive effects become more dominant. Moreover, the stress effect of CO_2 (increased blood corticoid levels) was found to be restricted to the period of uncompensated respiratory acidosis[14].

The stress resistance found in divers is in line with their subjective observations of increased "relaxation" in the course of prolonged diving training as instructors at the escape training tank.

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